



Proudly Presents the
Seminar Series:

Frontiers in Pharmacology

Richard Lewis, Ph.D.

Molecular and Cellular Physiology
Stanford Medical School

“The dynamic dyad: a mechanism for store-operated calcium signaling”

The activation of Ca^{2+} entry by the depletion of intracellular Ca^{2+} stores is a ubiquitous signaling mechanism with many important functions, including the activation of T lymphocytes by antigen. The underlying mechanism of this process has remained elusive for over 20 years, but the recent identification of the ER Ca^{2+} sensor (STIM1) and the CRAC channel pore subunit (Orai1) has enabled significant new insights into how depletion of Ca^{2+} in the ER opens Ca^{2+} channels in the plasma membrane. I will describe our recent studies supporting a new mechanism for store-operated Ca^{2+} signaling, in which store depletion causes STIM1 and Orai1 to move in a coordinated fashion to form closely apposed clusters in the ER and plasma membranes. The consequent opening of CRAC channels at these dyads represents the elementary unit of store-operated Ca^{2+} entry.

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10:00 am

Auditorium (Room 1005) in GBSF